Keith Arnold Reimer, M.D., Ph.D., Professor ofPathology at Duke University Medical School, internationallyrecognized cardiovascular scientist, pathologist, and teacher,died on March 15, 2002 of metastatic renal cell carcinoma atthe age of 56. Keith began his career in experimental pathol-ogy studying ischemic injury of the kidney, however he quickly-shifted his focus to myocardial ischemic injury, the field inwhich he went on to make his major scientific contributions.After completing the MD/PhD program at NorthwesternUniversity in Chicago, Keith joined the faculty at Duke Uni-versity in 1975 as Assistant Professor of Pathology. Early inhis career, working in collaboration with Dr. Robert B. Jen-nings, he published landmark studies describing and charac-terizing the “wavefront phenomenon” of myocardial ischemiccell death. These studies, published in two papers(Circulation 56: 786-794, 1977; and Laboratory Investigation40: 633-644, 1979), have been cited more than 1000 times.During the early 1980s, Keith developed methods to measurebaseline predictors of infarct size, such as area at risk andcollateral flow, that have become the standard for generatingreliable and reproducible data to test cardioprotective inter-ventions. The effort to discover cardioprotective interven-tions led to one of Keith’s most notable achievements — thedescription of one of the strongest and most reproducibleinterventions for reducing infarct size: ischemic precondition-ing. Numerous investigators and laboratories have worked tobetter understand this remarkably effective intervention, andthe ever-expanding number of studies on ischemic precondition-ing, in a wide variety of tissues, have consistently con-firmed the original observation that brief periods of ischemiaand reperfusion are not detrimental, but are actually marked-ly protective. The original article describing the phenomenonof ischemic preconditioning, “Preconditioning with ischemia:a delay of lethal cell injury in ischemic myocardium”(Circulation 74: 1124-1136, 1986) has been cited more than3700 times (the most cited paper in Circulation).

Keith was an active member of the ISHR since1976, and was elected a Councillor of the American Section in1979, serving until 1985. He was a finalist for the RichardBing Young Investigator Award of the ISHR in 1980. Keithserved as Secretary of the American Section from 1985-1994,and as a member of the Council of the International Societyfrom 1989-1995. In 1997, he became President-Elect of theAmerican Section and was the sitting President of the Ameri-can Section, as well as a member of the International ISHRCouncil, when he died.
Fabio Di Lisa, M.D.

Fabio Di Lisa is Professor of Biochemistry at the University of Padova, Italy. He received his MD degree and Board Certification in Cardiology at the Catholic University in Rome. Attracted by basic research more than clinical activities he joined Roberto Ferrari at the University of Parma to start experimental studies in the field of ischemia and reperfusion focusing on cardiac metabolism and mitochondrial function. This latter topic prompted his transfer to the University of Padova that was, and still is, the most advanced center for mitochondrial research in Italy. In 1987, accepting a NATO fellowship he worked in the laboratory of Loran Bieber at the Department of Biochemistry at Michigan State University focusing on the role carnitine in mitochondrial metabolism. He was then a Visiting Associate (1991-92) in the laboratory of Richard Hansford within the NIA-NIH Unit led by Edward Lakatta in Baltimore.

Prof. Di Lisa became a CNR Researcher in 1984 and then he was appointed Associate Professor and Full Professor of biochemistry in 1992 and 2002, respectively. In 1992 he started an independent laboratory in the Department of Biochemistry. Since 2007 he is affiliated with the Department of Biomedical Sciences. Besides an intense teaching schedule he has worked on numerous tasks coordinating the organization and the evaluation of scientific research both within the University of Padova and in National Councils.

Prof. Di Lisa was President of the European Section of the International Society for Heart Research (ES-ISHR) from 2005 to 2008. During this term, in 2007 he organized the ES-ISHR meeting in Padova and co-organized the ISHR World Congress in Bologna. He was elected Fellow of the ISHR in 2007, and at present, he is a member of the ISHR International Council. Regarding other Societies, he has been part of the Congress Programme Committee of the European Society of Cardiology for various terms (2005-2014), and he served on the Scientific Council of the Society for Heart and Vascular Metabolism organizing the annual meeting in Padova in 2009. Prof. Di Lisa is a member of the Editorial Board of the Journal of Biological Chemistry and journals in the cardiovascular field, such as the Journal of Molecular and Cellular Cardiology, Cardiovascular Research and Basic Research in Cardiology.

Prof. Di Lisa has provided significant contributions elucidating the role of mitochondrial dysfunction in cardiac diseases. He started his scientific activity characterizing mitochondrial alterations in ischemia/reperfusion and substrate utilization, especially highlighting the role of carnitine. Considering that findings obtained in isolated mitochondria might not always reflect the behavior of these organelles in situ, in the early nineties in Baltimore he started investigating mitochondrial function in isolated cardiomyocytes. He added the assessment of mitochondrial membrane potential to the measurement of intracellular and mitochondrial Ca\(^{2+}\) developed in Lakatta's laboratory to define patterns of mitochondrial dysfunction during anoxia and reoxygenation. In particular, he found that the mitochondrial membrane potential is maintained during anoxia using ATP produced by glycolysis, so that mitochondria changes from ATP producers into avid ATP utilisers. He also demonstrated that myocardial failure could be the result of a reduced Ca\(^{2+}\) uptake rather than Ca\(^{2+}\) overload. An interest in Ca\(^{2+}\) homeostasis triggered Di Lisa's interest in proteolysis of myofibrillar proteins. He demonstrated that calpain-catalyzed cleavage of troponin I and T is modulated by their phosphorylation, and their fragments are linked by transglutaminase as a result of Ca2+ overload occurring upon postischemic reperfusion. Myofibrillar proteins were also found to represent binding sites for cytosolic proteins redistributing during ischemia because of acidosis and ATP depletion.

A long-standing friendship with Paolo Bernardi was the driving force to start a fruitful collaboration on the mitochondrial permeability transition pore (PTP) that is still ongoing. By developing methods to study the PTP in isolated cells and intact hearts Prof. Di Lisa characterized the occurrence of transient and prolonged openings demonstrating that the latter modality is involved in cell death. In addition, PTP opening was causally related to NAD depletion and loss of viability induced by reperfusion. Derangements of mitochondria and myofibrillar proteins paved the way for studies on oxidative alterations and ROS formation. After highlighting troponymtin as a target of oxidative stress in reperfused hearts, in collaboration with Gerd Heusch and Rainer Schulz Prof. Di Lisa demonstrated that the oxidation of myofibrillar proteins correlates linearly with contractile impairment. This relationship that applies to various experimental models and human heart failure has been extended to muscular dystrophy. Concomitantly, bridging the gap between contractile proteins and mitochondria he provided evidence that reactive oxygen species are produced mostly within mitochondria, and especially by p66Shc and monoamine oxidases (MAO). Moving from ischemia/reperfusion injury to myocardial failure, in collaboration with Nazareno Paolocci and David Kass, MAO was shown to contribute to maladaptive remodeling highlighting also the potential therapeutic efficacy of MAO inhibition. This concept has been further documented in muscular dystrophy.

At present, the interest in PTP, ROS formation and Ca\(^{2+}\) homeostasis has been directed towards diabetic cardiomyopathy while maintaining the traditional focus on ischemia/reperfusion and mechanisms of cell death.

Previous Award Winners...

- Karin Sipido, MD, PhD: 2013
- Metin Avkiran, DSc, PhD: 2012
- Charles Murry, MD, PhD: 2011
- Richard Moss, PhD: 2010
- Elizabeth Murphy, PhD: 2009
- David Eisner, PhD: 2008
- Eduardo Marbán, MD: 2007
- Garrett Gross, PhD: 2006
- Masao Endoh, MD, PhD: 2005
- R. John Solaro, PhD: 2004
- Gerd Heusch, MD, PhD: 2003
- Roberto Bolli, MD: 2002